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# BEYOND HEMORRHAGE: FATAL SEVERE DENGUE WITH PARADOXICAL INFERIOR VENA CAVA THROMBOSIS AND CARDIAC DYSFUNCTION

General Medicine

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## ABSTRACT

Dengue infection is a major public health problem in tropical and subtropical countries. While most cases are self-limiting, a subset progresses to severe dengue with shock, multiorgan dysfunction, and atypical manifestations. We report a case of a 39-year-old female with severe dengue complicated by refractory shock, stress cardiomyopathy, and a paradoxical large inferior vena cava (IVC) thrombus extending into the right atrium. The case highlights the role of cytokine storm in the pathogenesis of severe dengue and underscores the need for high clinical suspicion for atypical manifestations.

## KEYWORDS

Dengue Shock Syndrome, cytokine storm, Dengue Inflammatory Syndrome, Inferior Vena Cava Thrombosis, Stress Cardiomyopathy, Myocarditis

## INTRODUCTION

Dengue fever, caused by the dengue virus (Flaviviridae), is the most prevalent arboviral infection worldwide, with India contributing a significant global burden. Clinical manifestations vary from mild febrile illness to severe dengue characterized by plasma leakage, shock, bleeding, and organ failure. Increasingly, atypical features such as myocarditis, cardiomyopathy, encephalopathy, and thrombotic events are being recognized. The hyperinflammatory state termed "dengue inflammatory syndrome" or cytokine storm is central to disease progression. We present a rare case of severe dengue with fulminant shock, paradoxical IVC thrombosis and stress cardiomyopathy, culminating in multiorgan failure and death.

### Case

A 39-year-old female of Korean descent presented with fever of one week duration, associated with chills, nausea, and generalized weakness. She had recently travelled from the UK to the USA and then to India. There was no prior comorbidity. She had a recent history of multiple episodes of syncope during this period of febrile illness, one episode of syncope was followed by a fall and head injury. She suffered a contused lacerated wound over her left parietal region that was sutured at a nearby hospital and a CT Brain was done that ruled out an intracranial bleed.

On admission, she was febrile with tachycardia (PR- 106 bpm) and borderline hypotension (100/60 mm Hg). Initial investigations revealed leukocytopenia (WBC 2720/µL), thrombocytopenia (27,000/µL), markedly elevated liver enzymes, hyponatremia (Na 125 mmol/L), and mild metabolic alkalosis. Renal function was preserved. Dengue NS1 antigen was positive, while malarial smear, rapid malaria antigen, Widal, TyphiDot, and influenza PCR were negative. (Table 1) She was given IV hydration, however, her blood pressure dropped to 70/50 mmHg, necessitating ICU transfer. Noradrenaline infusion and intravenous dexamethasone were initiated.

The following day, she had a precipitous drop in blood pressure (42/22 mmHg) with tachycardia (PR 130/min), cold clammy extremities, and acute breathlessness. She developed progressive thrombocytopenia (6000/µL), worsening transaminase elevation (AST- 388U/L, ALT-172U/L), hypoalbuminemia, and severe hyperferritinemia. Coagulation profile showed prolonged aPTT with normal fibrinogen levels.

ECG showed global ST-T elevation, suggestive of myocarditis. (Fig. 1) Cardiac biomarkers were elevated (HS-Troponin I: 1237.2 ng/L, NT-proBNP: 6704 pg/mL) suggestive of dengue myocarditis. Chest X-Ray was done that showed a faint right sided paracardiac shadow, likely due to pulmonary edema. (Fig. 2) Arterial and venous blood gases revealed worsening metabolic acidosis with lactate rising to 25 mmol/L. (Table 2)

**Table 1: Laboratory Tests:**

| Test | Values |
|---|---|
| CBC (WBC / Neutrophils / Platelets) | 14.4 / 2720 / 27000 |
| AST | 309 |
| ALT | 117 |
| GGT | 297 |
| Total Protein | 5.6 |
| Albumin | 3.3 |
| Serum Electrolytes (Na / K / Cl) | 125 / 4.4 / 91 |
| Creatinine | 0.5 |
| Cortisol | 11.4 |
| CRP | 7.6 |
| TFT - T3 | 56.6 |
| TFT - T4 | 6.11 |
| TFT - TSH | 3.22 |
| Plasma Fibrinogen | 226 |
| APTT | 54.3 |
| PT/INR | 11.2 / 1.07 |
| Triglycerides (TG) | 156 |
| HS Troponin I | 1237.2 |
| NT-pro BNP | 6704 |
| Ferritin | 6795 |

**Table 2: Serial VBGs**

| | | | | |
|---|---|---|---|---|
| VBG pH | 7.46 | 7.22 | 7.03 | 7.016 |
| VBG pCO2 | 30.4 | 44.6 | 51.2 | 37.2 |
| VBG Bicarbonate ($HCO_3^-$) | 21.3 | 15.5 | 11.2 | 9.1 |
| VBG Lactate | 2.4 | 6.5 | 23 | 25 |

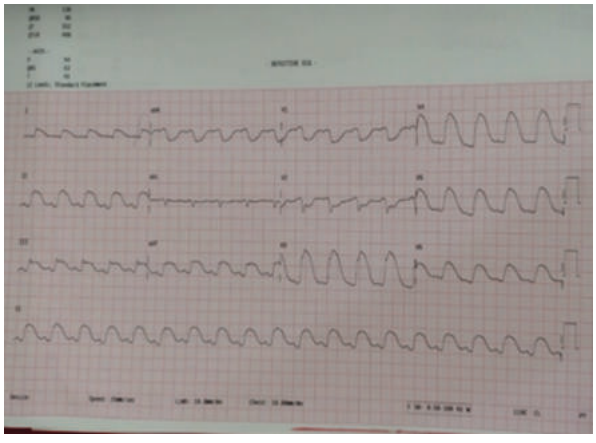

**Fig. 1. ECG s/o Evolving Myocarditis with Diffuse ST-T Changes**

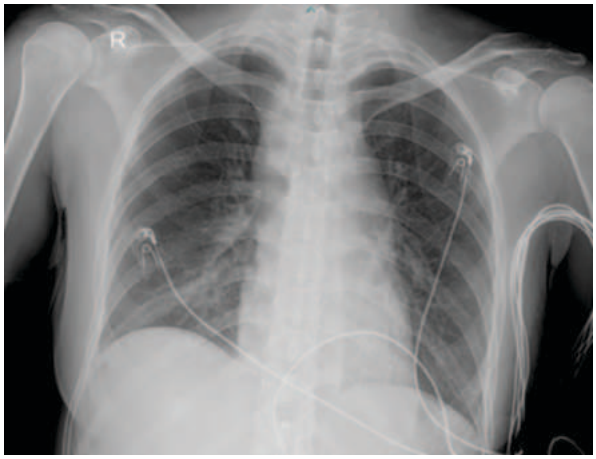

**Fig. 2. Chest XRay Suggestive of Faint Right Paracardiac Shadow**

She was intubated following gasping respirations and deteriorating mental status. VBG revealed severe lactic acidosis (pH 7.03, HCO3 11.2 mmol/L, lactate 23 mmol/L). Sodium bicarbonate infusion and aggressive fluid resuscitation were administered. She developed asystole, and cardiopulmonary resuscitation (CPR) was performed with return of spontaneous circulation (ROSC). 2D Echocardiography revealed severely reduced LVEF (15%), severe global hypokinesia, right atrial thrombus extending from the IVC (diameter 23 mm), dilated IVC with thrombus at its mouth, and PASP of 40 mmHg. The findings were consistent with stress cardiomyopathy and paradoxical IVC thrombosis. Despite escalation to triple vasopressor support, she sustained another cardiac arrest and could not be revived.

(Further evaluation of the IVC thrombus i.e. CT Venogram was planned but could not be done since the patient deteriorated within a few hours and could not be revived post the cardiac arrest.)

## DISCUSSION

Severe dengue is a hyperinflammatory state characterized by plasma leakage, shock, and multiorgan dysfunction. The index patient presented with severe dengue complicated by refractory shock, lactic acidosis, stress cardiomyopathy with myocarditis, and paradoxical IVC thrombus. While bleeding diathesis is classically associated with dengue, thrombotic complications are rarely reported, attributable to endothelial injury, immune-mediated platelet activation, and a paradoxical hypercoagulable state.

Thrombotic events in dengue are rare but are being increasingly recognized in clinical practice. A case report described a 16-year-old boy with dengue shock syndrome who developed ilio-femoral deep vein thrombosis during recovery, despite severe thrombocytopenia and no provoking risk factors. [1] Another report documented dengue shock syndrome complicated by acute liver failure, kidney injury, infective endocarditis, and deep vein thrombosis, while portal vein thrombosis has also been observed in severe dengue. [2, 3] These reports underscore that venous thrombosis, although uncommon, represents a serious complication of dengue.

Several mechanisms explain this paradoxical occurrence of thrombosis in the context of thrombocytopenia. Endothelial dysfunction, driven by dengue virus and its NS1 protein, contributes to vascular injury and exposes subendothelial procoagulant surfaces. Platelet dysfunction further adds to this milieu; platelets, although reduced, can form aggregates with monocytes and neutrophils, releasing inflammatory cytokines (IL-1β, IL-6, CXCL8) that promote microthrombosis. Simultaneously, activation of coagulation and fibrinolytic pathways can occur, resembling disseminated intravascular coagulation, favoring micro- and macrothrombus formation. [4] More recently, the concept of "thromboinflammation" has been emphasized, wherein the cytokine storm in dengue (notably TNF-α, IL-6, and IFN-γ) promotes tissue factor expression, reduces anticoagulant activity, and suppresses fibrinolysis, thereby creating a prothrombotic state. These mechanisms together can explain the development of extensive IVC thrombosis in this patient. [5]

Stress cardiomyopathy in dengue, although rare, has also been described and may mimic fulminant myocarditis. Proposed mechanisms include cytokine-mediated myocardial stunning, microvascular dysfunction, and catecholamine surge. [6] The echocardiographic finding of severe LV dysfunction (EF 15%) in our patient underscores the severity of cardiac involvement.

Hemophagocytic lymphohistiocytosis (HLH) is a rare but potentially fatal complication of dengue, characterized by an uncontrolled immune response leading to multiorgan damage. It presents with persistent high-grade fever, cytopenias, hepatosplenomegaly, hyperferritinemia, hypertriglyceridemia, and hemophagocytosis in bone marrow. In our patient, HLH was considered due to the presence of persistent fever, worsening cytopenias, and markedly elevated ferritin (6795 ng/mL). However, triglyceride levels were not significantly elevated (156 mg/dL), fibrinogen was within normal range (226 mg/dL), and bone marrow examination could not be performed before the patient's demise. Moreover, the rapid clinical deterioration with multiorgan failure and absence of definitive diagnostic features precluded a formal diagnosis. Thus, dengue-associated HLH remained a differential diagnosis but was ultimately ruled out based on insufficient criteria and lack of confirmatory evidence. Nevertheless, this highlights the diagnostic challenge of distinguishing HLH from cytokine storm in severe dengue, as both share overlapping features. [7]

This case emphasizes that severe dengue is not solely a hemorrhagic disease but a multisystem hyperinflammatory disorder with protean manifestations, including thrombotic events and cardiomyopathy. Early suspicion and timely intervention may improve outcomes, but fulminant cases such as this remain challenging.

## CONCLUSION

This case highlights the rare occurrence of IVC thrombus in severe dengue complicated by cytokine storm and dengue inflammatory syndrome. Clinicians should remain vigilant for atypical and hyperinflammatory manifestations of dengue, as early recognition may guide timely interventions. Further research is needed to establish the role of immunomodulatory therapies in cytokine storm–driven severe dengue.

## REFERENCES


(1) Ranasinghe KMIU, Dissanayaka D, Thirumavalavan K, Wijesinghe P, Samarasekara W. An unusual case of dengue shock syndrome complicated by ilio-femoral deep vein thrombosis. BMC Infect Dis. 2020;20:335.
(2) Chlebicki MP, Ang B. Dengue hemorrhagic fever complicated by deep vein thrombosis and pulmonary embolism. J Infect. 2005;51(6):e243–6.
(3) Ralapanawa DM, Kularatne SA, Jayalath WA. Dengue hemorrhagic fever complicated with acute liver failure, encephalopathy and disseminated intravascular coagulation: a case report. J Med Case Rep. 2016;10:260.
(4) Wills BA, Oragui EE, Stephens AC, Daramola OA, Dung NM, Loan HT, et al. Coagulation abnormalities in dengue hemorrhagic fever: serial investigations in 167 Vietnamese children with dengue shock syndrome. Clin Infect Dis. 2002;35(3):277–85.
(5) Tan LK, Tan CH, Tang KF. The role of cytokines in pathogenesis of dengue infection. Curr Opin Infect Dis. 2016;29(4):347–52.
(6) Gupta VK, Gadpayle AK. Subclinical cardiac involvement in dengue haemorrhagic fever. J Indian Acad Clin Med. 2010;11(2):107–11.
(7) Rueda M, Yoo M, Polanco CM, Mejia M, Jaramillo J, Villar LA. Dengue-associated hemophagocytic lymphohistiocytosis: a systematic review of case reports. Rev Inst Med Trop Sao Paulo. 2019;61:e49.